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Case report

A fatally mistaken fruit juice drink: An unordinary way of cocaine intoxication

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ABSTRACT

Cocaine is one of the drugs of abuse more frequently consumed in Spain. Furthermore, Spain due to its geographical position is used by trafficker's organizations as the port of entrance of cocaine in the European Union. We present here a case of a fatal intoxication caused by a mistake in the cocaine distribution net in our country. Cocaine was concealed in a tropical juice only sold by the Internet.

1. Introduction

According to data released by the European Monitoring Centre for Drugs and Drug Addiction,[1] an increase in cocaine consumption has been recorded in Spain. In the last few years, cocaine has emerged as a major cause of morbidity and mortality, being cocaine consumption one of the issues that arises higher concern in our society. Nowadays, cocaine-related death is a serious and possibly increasing problem in European countries. Despite the limitations of the available data, cocaine seems to have played a determinant role in 1–15% of drug-related deaths reported by the European Monitoring Centre for Drugs and Drug Addiction.[1] Furthermore, due to its geographical position organized drug traffickers use Spain as the drug port of entrance in the European Union.

Except in the case of drug couriers ("body packers") with massive drug exposure, death is not dose related, and cocaine blood levels cannot be used to predict toxicity.[2] Cocaine-related deaths occur for the major part after prolonged drugs use and mere presence of cocaine in fluids or tissues does not prove that death was due to cocaine consumption. The aim of the present paper is to present a curious case of fatal intoxication of cocaine erroneously consumed.

2. Case report

2.1. Anamnestic data

A 40-year-old male was told by some friends that juice of a tropical fruit called Noni was a good way of enhancing energy and wellbeing. So, he ordered by Internet through a Mexican distributor three bottles of the fruit juice.

As his partner told the police, once the bottles arrived, she opened one of them and served him. Suddenly after drinking, he felt very bad, falling on the floor. She also stated that she broke the bottle she still had in her hands.

He presented the following signs and symptoms: diplopia, xerostomia, loss of balance, ataxia, vomits and convulsions. His partner called urgent medical assistance. Although cardiopulmonary resuscitation was applied he finally died due to a cardiorespiratory arrest only 30 min after drinking the fruit juice.

No former drug consumption was reported by family or suspected by coroner at scene investigation. Nevertheless, in order to discard intoxication, coroner sent us femoral blood, urine and vitreous humour from the deceased. Also, two bottles of Thaitian Noni fruit juice found at the scene were sent to our laboratory. Bottles were identified, at random, as number one and two, respectively. Bottle number 1 was filled with a dark green liquid. Bottle number 2 was filled with a white solid suspended in a clear liquid.

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2.2. Materials

Reactives and solvents used were of analytical grade (Merck, Barcelona, Spain) and analytical standards were purchased from Promochem (Austin, TX, USA). Blood and urine specimens were collected by the coroner and kept in tubes with sodium fluoride as a preservative and potassium oxalate as anticoagulant. On their arrival to the laboratory, all specimens were stored at 4 °C until their extraction.

2.3. Analytical methods

Ethanol was analysed by means of headspace GC-FID, according to the Spanish Official Method for ethanol analysis in blood samples.[3]

Screening of cocaine metabolite (benzoylecgonine) was performed by means of homogeneous enzyme immunoassay CEDIA® according to manufacturers' instructions in the case of urine. Blood samples were deproteinized with acetone prior to CEDIA® analysis.[4]

All specimens were extracted by means of SPE (Bond-Elut® certified for basic compounds Varian Harbour City, CA, USA), following the normal procedure in our laboratory. Preliminary analyte indentification was performed by means of gas chromatography, using a Varian CP-3800 (Walnut Creek, CA, USA) gas chromatograph fitted with a NPD detector. The column used was a fused-silica Ultra-1 column (200 μm × 0.33 mm × 25 m) coated with methyl siloxane. Carrier gas was helium at a rate of 1.7 mL/min. Injector and detector temperatures were 280 °C and 300 °C respectively. Initial oven temperature was 60 °C, maintained for 2 min, increasing at 12 °C/min to 290 °C staying at this temperature for 10 min. Injection volume was 1 μL.

The presence of cocaine and its metabolites (ethylbenzoylecgonine, benzoylecgonine and methylecgonine) was confirmed and quantitated by GC–MS. Dry residues were heated 20 min at 70 °C with 50 μL of the derivatization reagent, BSTFA (N, O-bis (trimethylsilyl) trifluoroacetamide).[5] A Hewlett–Packard (Agilent Technologies, Palo Alto, CA, USA) gas chromatograph Model 5890 II equipped with a MS detector HP 5973 working in electron impact (EI) mode under selected ion monitoring (SIM) conditions. The column used was a fused-silica capillary Ultra-1 column (200 μm × 0.33 mm × 25 m) coated with methyl siloxane. Carrier gas was helium at a rate of 1.7 mL/min. Injector temperature was 280 °C. MS source and MS quadrupole temperatures were 230 °C and 250 °C, respectively. Initial oven temperature was 60 °C, maintained for 3 min, increasing at 12 °C/min to 290 °C staying at this temperature for 10 min. Injection volume was 1 μL. Analytical method has been fully validated previously.[5]

2.4. Toxicological results

The analysis of the bottles of tropical fruit juice yielded the results that are shown in Table 1. Although, ethanol was detected in both bottles, cocaine and related products were only detected in bottle number two. It is the first time, to our knowledge, that cocaethylene formation in vitro by a non-enzymatic pathway is reported.

Table 1
Results obtained in the analysis of Noni® bottles (g/L).

	Bottle 1	Bottle 2
Ethanol	0.14	5.45
Cocaine	<LOD[a]	24.17
Benzoylecgonine	<LOD	0.71
Methylecgonine	<LOD	0.28
Cocaethylene	<LOD	0.09

[a] <LOD: lower than limit of detection.

Table 2
Cocaine and its metabolites concentrations in biological fluids analysed from the deceased (mg/L).

	Blood	Urine	Vitreous humour
Cocaine	2.27	15.51	4.55
Benzoylecgonine	5.31	25.31	3.85
Methylecgonine	14.74	14.06	4.47

No ethanol was detected in blood. Cocaine, methylecgonine and benzoylecgonine were detected in blood, urine and vitreous humour, as it is shown in Table 2. No other toxic compounds were detected.

2.5. Autopsy findings and histological examinations

Coroner suspected that the cause of death could be a vasovagal syncope due to the fact that the deceased could have drunk a very cold drink or perhaps some kind of allergic response. Autopsy revealed non-specific signs such as viscera congestion and pulmonary edema. Specimens of heart, lung, brain, liver, spleen, kidney fixed in fomaline were received.

Macroscopic and histological examinations were applied to the samples received. After being embedded in paraffin, specimens were sectioned at 5 μm and stained with hematoxylin-eosin and with Masson Trichrome.

Heart was weighted, opened and inspected and any gross changes examined histologically. The coronary arteries were cross-sectioned at 3 mm intervals and any segment with luminal modification was processed for histology. Heart was macroscopically normal and weighted 340 g. Right and left ventricular wall thicknesses were of 0.2 cm and 1.4 cm, respectively. The myocardium showed a greyish coloration and an elastic consistency. The histological examination revealed a mild (40%) luminal stenosis degree in right and left coronary arteries. Furthermore, myocardiocytes with signs of recent necrosis were observed. Those signs were coagulative myocytolysis with hypercontraction bands, loss of the nuclei and vacuolized cytoplasma. However, no inflammatory cell infiltration was present.

A fragment of brain (13 × 11 × 5 cm) was received. Its surface was palid and soft. Vascular congestion and some haemorraghic foci are observed in the encephalic parenchyma.

Five fragments of lung were received. The outer surface was brown-reddish and presented an elastic consistency. Lung parenchyma was partially emphysematous. Bonchioli ducts and alveoli lumen showed the presence of aspirated material from gastric contents. Edema liquid is observed inside alveoli. Vascular congestion and some haemorraghic foci are observed.

A fragment of liver (14 × 5 × 2 cm) with a smooth and greenish outer surface was received. Some necrotic hepatocytes near centrilobular veins were observed. There were some intraparenchymal necrotic foci and microvesicular steatosis.

Therefore, histological findings in heart and liver were suggestive of acute necrosis of toxic etiology. Other organs were unremarkable on both gross and microscopic examination.

3. Discussion

We present here a fatal case of an accidental cocaine overdose. The toxicological examination revealed the presence of cocaine in blood in a concentration of 2.27 mg/L (Table 2). Unfortunately, cocaine intake could not be established because the open bottle was broken and gastric contents were not submitted to our laboratory.

To assess the relevance of the obtained results, we compared them with other fatal cases reported. The literature offers

considerable toxicological data about cocaine-related deaths, and it is clear that correlation of a specific blood or tissue concentration with toxicity is not generally possible. For some authors, establishing the presence of cocaine in the blood, in the absence of other findings, may signify death by cocaine poisoning. Due to the fact that acute cardiac effects of cocaine are independent of the cocaine concentration in the blood, establishing the presence of cocaine in the blood, in the absence of other findings, can allow a death to be certified as resulting from cocaine intoxication. However, others would undoubtedly insist that isolated cocaine levels cannot be used to explain the cause of death. In fact the presence of low levels of cocaine is proof only of cocaine use, if the appropriate anatomical or histological changes are present, cocaine may be the cause of death even if it is not detectable in the blood.[6–10]

Knowledge of the anamnestic data, the results of a complete scene investigation, toxicological analysis and an understanding of the current relevant forensic literature on this subject should be available prior to any interpretation of the significance of cocaine upon a specific death.

The interpretation of postmortem blood concentrations is even more complicated than attempts at making such correlations in the living. Before the current cocaine pandemic, blood concentrations of more than 5 mg/L were thought to be uniformly fatal. With more experience, it has become apparent that isolated postmortem blood concentrations cannot be used to determine the cause of death. Tolerance on a massive scale occurs and concentrations well in excess of 5 mg/L can be encountered in cases of trauma death where the presence of cocaine is clearly an unrelated finding.[6,11] However, some authors have stated that cocaine concentration in excess of 2 mg/L, as the one reported here, may suggest an elevated dosing.[12]

It is generally known that cocaine causes serious systemic injuries. Coagulative myocytolysis is observed in many humans and in experimental conditions, including ischaemic heart disease. However, several facts support the view that coagulative myocytolysis is due to catecholamine cardiotoxicity which can be reproduced experimentally. This toxicity seems to be due to an excess of intramyocellular Ca^{2+} cell influx rather than coronary spam and/or increased oxygen demand of myocells.[13]

Numerous reports attest cocaine hepatotoxic potential in humans. Silva et al.[14] showed that a 60% of patients who suffered a cocaine acute intoxication presented some kind of hepatic injury. Severe injuries are characterized by coagulation necrosis involving not only perivenular but also mid-zones and microvesicular steatosis. While the perivenular zone necrosis might be ascribed to hypoxia, the microvesicular steatosis is presumably cocaine-induced.[15]

Therefore, in this case death was attributed to an acute cocaine intoxication due to the high blood cocaine concentration measured and the facts that the deceased was not a regular cocaine consumer, no other toxicologically relevant substances were detected in biological fluids analysed, the death occurred suddenly just after drinking Noni juice and the histological findings of necrotic foci in liver and heart concordant of a acute toxic damage.

4. Conclusions

We emphasize the importance of toxicological examination in every sudden unexpected death of people during the process of medico-legal investigation. To our knowledge, this is the first time that cocaine concealed in this kind of tropical fruit juice is reported. As it is widely known, buying medicines, drinks and food in the Internet presents a potential danger to Public Health. Furthermore, Internet trading is frequently uncontrolled by Public Health authorities giving sometimes opportunities to criminal organizations to traffic with drugs of abuse.

The conclusion that cocaine is directly responsible for the immediate cause of death should be considered only when there is a reasonably understanding of the circumstances or facts surrounding the death. In order to reach such conclusion, another more obvious and immediate cause of death must be absent, or, at least cocaine must be shown to be a significant contributing factor in the chain of medical findings that lead directly to the immediate cause of death. Due to the facts discussed above, manner and cause of death were reported as accidental fatal intoxication by cocaine.

Finally, we can state that a diversion in the distribution net of drug traffickers leaded to fatal consequences in an innocent person.

Conflict of interest

All authors have made a significant contribution to the findings and methods in the paper. There are no financial or commercial interests. The work has not been published and has not been submitted simultaneously to any other journal.

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